CHAPTER 16
Abnormalities of Pulse Rhythm

KEY TEACHING POINTS

- There are five basic abnormalities of the pulse rhythm: the pause, regular bradycardia, regular tachycardia, irregular rhythm varying with respiration, and chaotic rhythm (irregularly irregular rhythm).
- The pause is caused by premature beats or second-degree heart block. It is named by the number of regular beats before each pause (i.e., bigeminal, trigeminal, or group beating).
- Regular bradycardia is caused by sinus bradycardia, complete heart block, or halved pulse.
- Regular tachycardia is caused by sinus tachycardia, atrial flutter, paroxysmal supraventricular tachycardia, or ventricular tachycardia.
- An irregular rhythm varying with respiration is sinus arrhythmia, a common rhythm of young, healthy patients.
- The chaotic rhythm is caused by atrial fibrillation or multifocal extrasystoles.
- These arrhythmias may be distinguished by examination of the venous waveforms, heart tones, and response to vagal maneuvers. Even so, all arrhythmias require electrocardiography for confirmation and monitoring.

I. INTRODUCTION

In the late 19th and early 20th centuries, before the introduction of electrocardiography, clinicians could examine the patient's arterial pulse, heart tones, and jugular venous waveforms and, from these observations alone, diagnose atrial and ventricular premature contractions, atrial flutter, atrial fibrillation, complete heart block, Mobitz 1 and 2 atrioventricular block, and sinoatrial block.[1-3] In fact, clinicians were familiar enough with the bedside findings of these arrhythmias that early textbooks of electrocardiography included tracings of the arterial and venous pulse to help explain the electrocardiogram (ECG; Fig. 16.1).[4]

The bedside diagnosis of arrhythmias today is probably little more than a intellectual game, because all significant arrhythmias require electrocardiography for confirmation and monitoring. Nonetheless, bedside diagnosis of arrhythmias is still possible, using principles discovered 100 years ago by Mackenzie, Wenckebach, and Lewis. These principles, based on extensive investigation and many polygraph recordings of the arterial and venous pulse,[1-4] allow diagnosis of simple arrhythmias when the electrocardiograph is not immediately nearby.

II. TECHNIQUE

The first step in diagnosing arrhythmias is to determine the basic rhythm of the patient's radial pulse. Most arrhythmias can be classified into one of five basic

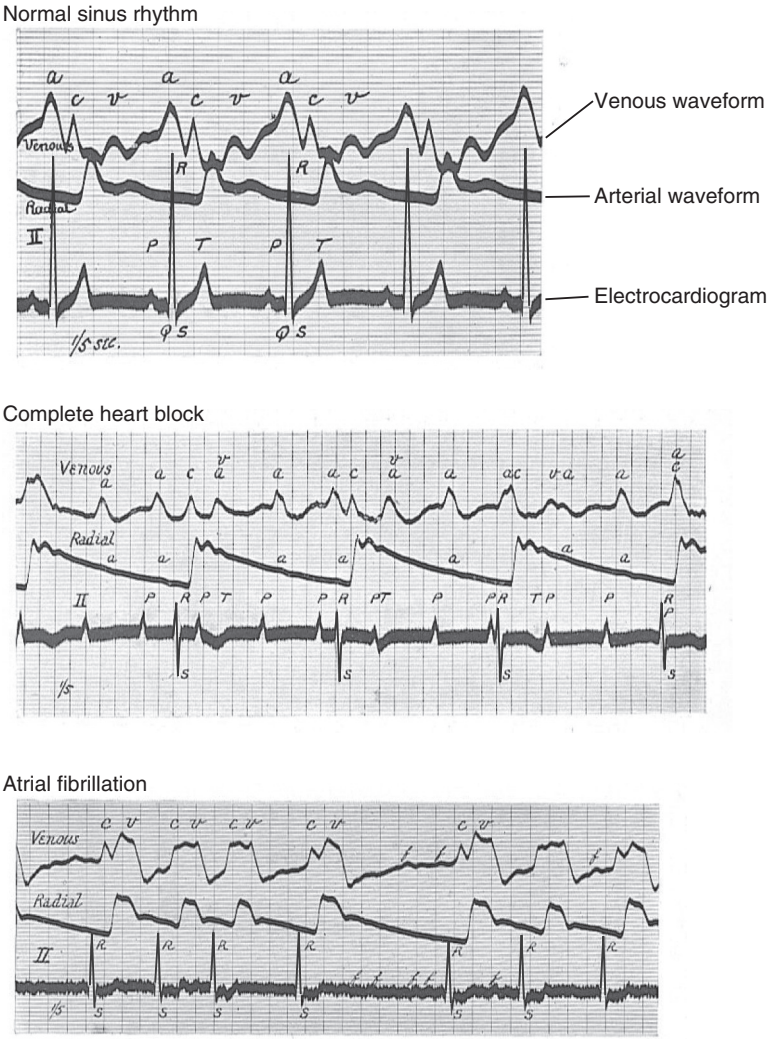

FIG. 16.1 SIMULTANEOUS VENOUS, ARTERIAL, AND ELECTROCARDIOGRAPHIC CURVES. To help clinicians understand the P, QRS, and T waves of the newly introduced electrocardiogram, early textbooks displayed simultaneous venous and arterial waveforms with the electrocardiogram. These examples, reproduced from Sir Thomas Lewis's 1925 *Mechanism and Graphic Registration of the Heart Beat*,[4] depict normal sinus rhythm (*top*), complete heart block (*middle*), and atrial fibrillation (*bottom*) (see the text).

abnormalities: (1) the pause, (2) regular bradycardia, (3) regular tachycardia, (4) irregular rhythm that varies with respiration, and (5) irregularly irregular (or chaotic) rhythm (Fig. 16.2).

The radial pulse may not correspond to the ventricular pulse (or apical pulse), as determined by auscultation of the heart tones or palpation of the cardiac impulse,

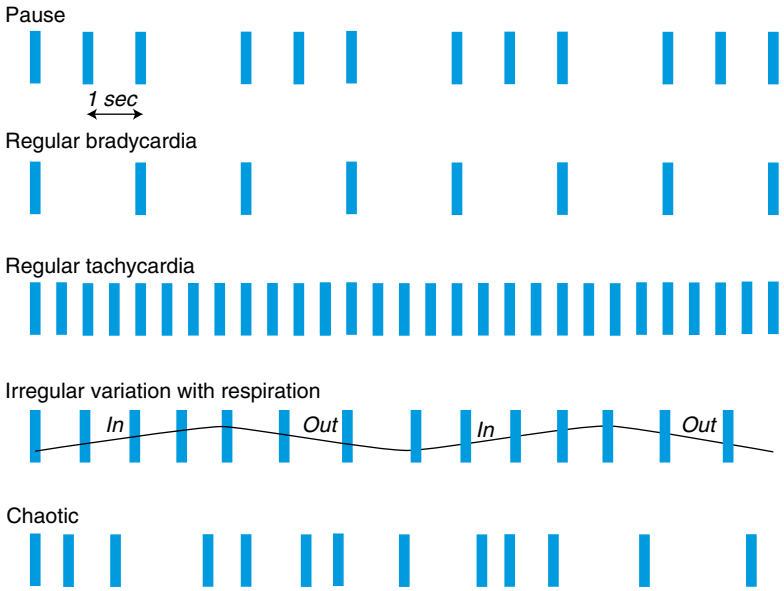

FIG. 16.2 BASIC ABNORMALITIES OF PULSE RHYTHM. Basic abnormalities include (1) the pause, (2) regular bradycardia, (3) regular tachycardia, (4) irregular rhythm that varies with respiration ("in" depicts inspiration and "out" depicts expiration), and (5) irregularly irregular (or "chaotic") rhythm. See the text.

because some ventricular contractions are too weak to propel blood to the radial artery. Although the clinician must compare the radial pulse with the ventricular pulse to diagnose arrhythmias, the difference in *rate* between the two by itself indicates no particular diagnosis.

After the basic rhythm of the radial pulse is identified, analysis of the jugular venous waveforms, heart tones, and response of the heart rhythm to vagal maneuvers may further distinguish the various causes.

III. THE FINDINGS AND THEIR CLINICAL SIGNIFICANCE

A. THE PAUSE

The pause has two important causes: premature contractions (common) and heart block (uncommon).

1. TERMINOLOGY

When the radial pulse consists of the regular repetition of two beats followed by a pause, the term **bigeminal pulse** or **bigeminal rhythm** is used. When there are three radial pulse beats between each pause, the appropriate term is **trigeminal pulse** or **trigeminal rhythm**. The finding of several beats between each pause is usually called **group beating**, and even longer periods of regular rhythm interrupted by the rare pause are sometimes referred to as **pulse intermissions**. The basic mechanism for

all these rhythm disturbances is the same; only the frequency of premature beats or heart block differs among them.

Because the cadence of these rhythms becomes predictable after short periods of observation, the term *regularly irregular* is sometimes used. This term, however, inaccurately conveys to others what is actually going on and is best discarded.

2. BASIC MECHANISM OF THE PAUSE

The pause has three basic mechanisms, illustrated in Fig. 16.3. The two most important questions that distinguish these mechanisms are the following: (1) Is there a premature radial pulse immediately preceding the pause? (2) Do additional ventricular beats (identified by listening to the heart tones or palpating the apical pulse) occur during the pause?

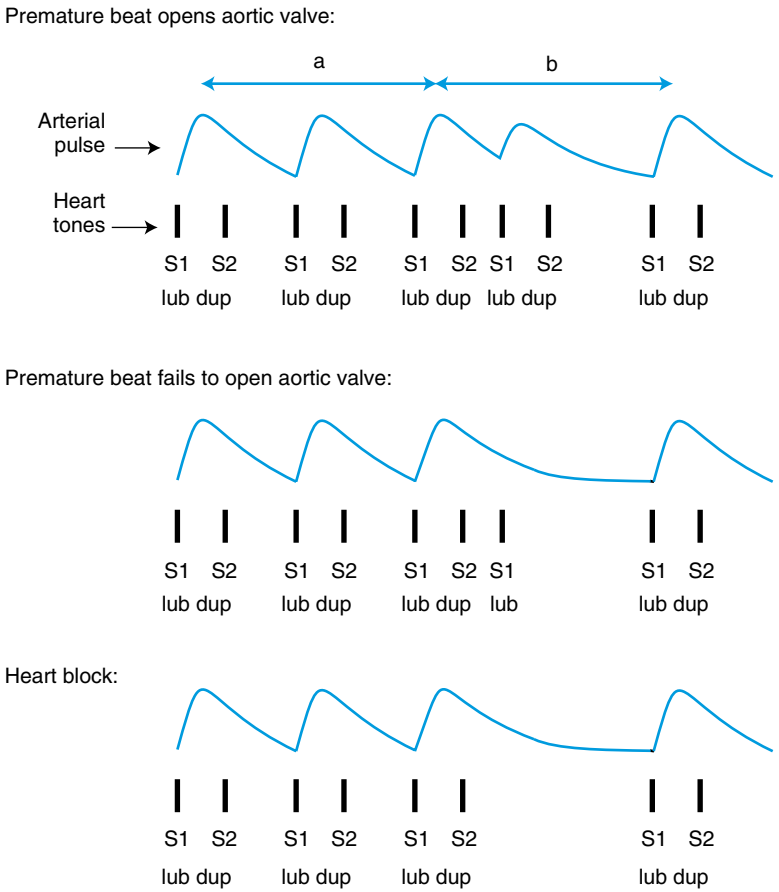

FIG. 16.3 MECHANISM OF THE PAUSE. The radial pulse tracing and heart tones are presented, illustrating the three mechanisms for the pause: (1) premature contraction that opens the aortic valve, (2) premature contraction that fails to open the aortic valve, and (3) heart block. Onomatopoeia of the heart tones appears below each tracing. ("lub" is the first heart sound; "dup" is the second heart sound). See the text.

A. PREMATURE BEAT

Patients with premature contractions (the first two examples in Fig. 16.3) have evidence of a premature ventricular beat during or immediately preceding the pause in the radial pulse. This early beat is always evident in the form of a palpable apical impulse or additional heart tones, although it may not be felt in the radial artery.

Some premature contractions are strong enough to open the aortic valve (first example in Fig. 16.3). If so, the clinician will feel a quick beat in the radial pulse just preceding the pause, although the quick beat is usually not as strong as a normal sinus beat. When listening to the heart tones, the clinician will hear both the first and second heart sounds of the early beat, which produces the following characteristic cadence:

lub dup lub dup lub dup lub dup lub dup

(In this and the following two examples, *lub* is the first heart sound and *dup* is the second sound; each rhythm begins with three normal beats, i.e., three *lub dups*.)

If the premature contraction is too weak to open the aortic valve (second example in Fig. 16.3), the clinician palpating the pulse will not detect the quick beat but only feel the pause. Listening to the heart, he or she will only hear the first sound of the premature beat (S_2 is absent because the aortic valve never opens):

lub dup lub dup lub dup lub lub dup

B. HEART BLOCK

Patients with heart block (third example in Fig. 16.3), whether sinoatrial or atrioventricular, lack a palpable apical impulse or extra heart tones during the pause. The cadence of heart tones contrasts with those of the premature beat:

lub dup lub dup lub dup lub dup

3. BIGEMINAL AND TRIGEMINAL RHYTHMS, AND GROUPED BEATING

Based upon the mechanisms previously discussed, there are three causes of the bigeminal pulse rhythm: (1) alternating normal and premature contractions; (2) premature contractions occurring every third beat, although the premature contraction is too weak to open the aortic valve; and (3) 3:2 heart block (atrioventricular or sinoatrial). In causes 2 and 3, both beats of the couplet are strong, but cause 2 has evidence of a ventricular contraction during the pause, whereas cause 3 does not.

The same analysis is used for trigeminal rhythms and grouped beating (i.e., in trigeminal rhythms, possible causes are premature contractions after every two or three normal beats or 4:3 heart block).

4. ATRIAL VERSUS VENTRICULAR PREMATURE CONTRACTIONS

Two helpful bedside findings distinguish atrial premature contractions from ventricular ones:

A. COMPENSATORY PAUSE

Beats that originate in the ventricle usually do not upset the underlying sinus rhythm, causing the beat immediately following the pause to fall exactly when the clinician anticipates it. Tapping the foot during the normal regular rhythm helps determine this. In Fig. 16.3, the distance "b" equals "a," meaning there is a "complete compensatory pause."

Beats that originate in the atria, in contrast, often reset the sinus node, causing the next beat to appear earlier than expected. In Fig. 16.3, "b" would be less than "a," and the clinician tapping the foot would find that the basic meter of rhythm changes.

This rule is more helpful when the pause is not compensatory (i.e., b < a, indicating the beat is atrial), because many atrial premature contractions also seem to have a complete compensatory pause at the bedside.

B. CANNON A WAVES

The appearance of a sudden prominent venous wave in the neck (cannon A wave) *during the pause* indicates that the premature beat was ventricular (see also Chapter 36). This occurs because the right atrium, still beating under the direction of the uninterrupted sinus impulses, contracts after the ventricular premature contraction has closed the tricuspid valve. Rarely, extremely premature ectopic atrial beats may also produce cannon A waves, but these waves precede the first heart sound of the premature contraction, whereas cannon A waves from ventricular premature contractions always follow the first heart sound of the premature beat.

B. REGULAR BRADYCARDIA

Regular bradycardia is a heart rate of less than 50 beats/minute. There are three causes of regular bradycardia that are recognizable at the bedside: sinus bradycardia, complete heart block, and halved pulse.

1. SINUS BRADYCARDIA

This arrhythmia resembles the normal rhythm in every way except for the abnormally slow rate: the venous waveforms in the neck are normal, the intensity of the first heart sound is the same with each beat, and there is no evidence of ventricular contractions between radial pulsations (as determined by palpation of apical impulse or auscultation of the heart tones).

2. COMPLETE HEART BLOCK

In complete heart block, the atria and ventricles beat independently of each other (i.e., atrioventricular dissociation). Sometimes the atrial and ventricular contractions are contiguous, and sometimes they are far apart. Atrioventricular dissociation causes two important bedside findings: changing intensity of the first heart sound and intermittent cannon A waves in the venous pulse.

A. CHANGING INTENSITY OF THE FIRST HEART SOUND

In complete heart block, the first heart sound of most beats is faint. Intermittently, however, the atrium contracts just before the ventricle contraction, which results in a first heart sound of booming intensity (named **bruit de canon** because of its explosive quality; see Chapter 40 for the pathophysiology of S_1 intensity).[5]

The finding of a changing first heart sound is only significant when the pulse is regular, because in irregular rhythms its intensity naturally varies with the length of the previous diastole (i.e., long diastoles intensify the first heart sound of the next beat; short diastoles diminish it). If the ventricular pulse is regular, however,

a changing intensity of the first heart sound (or intermittent "booming" of the first heart sound) indicates only one diagnosis, atrioventricular dissociation.

B. INTERMITTENT APPEARANCE OF CANNON A WAVES IN THE VENOUS PULSE

In complete heart block, when an atrial contraction falls intermittently just after a ventricular contraction, the right atrium will contract against a closed tricuspid valve, causing an abrupt systolic outward wave in the jugular venous pulse (i.e., cannon A wave; see also Chapter 36).

In many different arrhythmias, cannon A waves appear with *every* arterial pulse. If cannon A waves appear *intermittently*, however, in a patient whose ventricular pulse is *regular*, the only possible diagnosis is atrioventricular dissociation.

C. OTHER EVIDENCE OF ATRIOVENTRICULAR DISSOCIATION

Other uncommon signs of atrioventricular dissociation are regular small A waves in the venous pulse; regular muffled fourth heart sounds at the apex; or in patients with mitral stenosis, regular short murmurs from the atrium pushing blood across the stenotic valve. All of these findings represent regular atrial contractions that continue during the long ventricular diastoles.

A rare sign of complete heart block is an intermittently audible summation gallop (or third heart sound; see Chapter 41).[6]

3. HALVED PULSE

Halved pulse refers to the finding of twice as many ventricular beats as radial pulse beats. This is usually due to premature contractions that appear every other beat but are too weak to open the aortic valve and reach the radial pulse. Rarely, pulsus alternans may be the cause (**total alternans**),[7] although in these patients, the heart tones at the apex are regular, whereas in premature contractions, they are bigeminal.

C. REGULAR TACHYCARDIA

The regular tachycardias that *sometimes* are recognizable at the bedside include sinus tachycardia, atrial flutter, paroxysmal supraventricular tachycardia, and ventricular tachycardia. The bedside observations that distinguish these arrhythmias are response to vagal maneuvers, signs of atrioventricular dissociation, and abnormalities of the neck veins. Even so, bedside examination is diagnostic in only the minority of patients with rapid rates, and the careful clinician always relies on electrocardiography for diagnosis.

1. VAGAL MANEUVERS

The usual maneuvers are the Valsalva maneuver and carotid artery massage.

A. TECHNIQUE

Both maneuvers are performed when the patient is supine. To perform the Valsalva maneuver, the clinician asks the patient to bear down and strain against a closed glottis as if "having a bowel movement." Patients who have difficulty following this instruction sometimes respond better when asked to put the tip of their own thumb into their mouth and pretend it is a balloon to blow up. In patients with supraventricular tachycardia, 15 seconds of straining is as effective as 30 seconds.[8] The Valsalva maneuver increases vagal tone and has its maximal effect on tachycardias *after* the release of the Valsalva, not while the patient is straining.[8]

In carotid artery massage, the clinician finds the bifurcation of one carotid artery, located just below the angle of the jaw, and massages or presses on it for 5 seconds.[8,9]

The Valsalva maneuver is preferred for two reasons: (1) It tends to be more efficacious, terminating supraventricular tachycardia 20% to 50% of the time, compared with only a 10% efficacy using carotid massage;[8,10] and (2) in elderly patients with carotid artery disease, carotid artery massage may cause a stroke.[11,12]

B. RESPONSE OF REGULAR TACHYCARDIAS TO VAGAL MANEUVERS[9]

Transient slowing of the pulse during a vagal maneuver indicates sinus tachycardia. **Abrupt termination** of the tachycardia indicates paroxysmal supraventricular tachycardia (which occurs with both nodal reentry tachycardias and reciprocating tachycardias from accessory pathways). **Abrupt halving** of the rate may occur in atrial flutter. **No response** is unhelpful, being characteristic of ventricular tachycardia[13] but also occurring with every other regular tachycardia.[8,10]

2. ATRIOVENTRICULAR DISSOCIATION

Any finding of atrioventricular dissociation in patients with regular tachycardia indicates the rhythm is ventricular tachycardia. These findings include the *intermittent* appearance of cannon A waves in the neck veins, changing intensity of the first heart sound, and changing systolic blood pressure (usually detected with the blood pressure cuff).[14] In one study of patients with ventricular tachycardia, in which atrioventricular association or dissociation was determined by pacing (EBM Box 16.1), the finding of a changing S_1 increased probability of atrioventricular dissociation (likelihood ratio [LR] = 24.4) and the *absence* of intermittent cannon A waves decreased probability of atrioventricular dissociation (LR = 0.1).

Even so, these LRs are misleading because some patients with ventricular tachycardia lack atrioventricular dissociation and instead have 1:1 retrograde conduction or atrial fibrillation.[13] Given the serious consequences of misdiagnosing the regular tachycardia rhythm, an ECG should always be obtained.

3. FLUTTER WAVES IN THE VENOUS PULSE

In elderly patients with a ventricular pulse of 130 to 160 beats/minute, the clinician should suspect atrial flutter with 2:1 conduction. In addition to performing vagal maneuvers, the clinician may see rapid, small undulations (with a rate about 300/minute) in the venous pulse, which are called flutter waves (or f waves) and correspond to the wave of the same name on the ECG.[16]

4. SENSATION OF POUNDING IN THE NECK

A common cause of regular tachycardia is atrioventricular nodal reentrant tachycardias. In patients with this arrhythmia, the retrograde P wave of every beat coincides with the QRS complex, resulting in simultaneous cannon A venous pulsations and carotid arterial pulsations in the neck of affected patients, thus creating conspicuous pounding neck palpitations. Other causes of regular tachycardias are less likely to create neck palpitations because the atrial and ventricular contractions occur at slightly different times. (In patients with reciprocating tachycardias from accessory pathways, for example, the atrial contraction occurs after the ventricular contraction.)

In studies of patients referred to electrophysiology specialists because of intermittent rapid palpitations, the symptom of *rapid, regular* pounding in the neck during the palpitations distinguished atrioventricular nodal reentrant tachycardia from other causes of tachycardia with a sensitivity of 20% to 92%, specificity of 83% to 100%, positive LR of 9.6, and negative LR of 0.5.[17-19]

EBM BOX 16.1
*Atrioventricular Dissociation and Ventricular Tachycardia**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Varying arterial pulse[15]	63	70	NS	NS
Intermittent cannon A waves, neck veins[15]	96	75	**3.8**	**0.1**
Changing intensity S_1[15]	58	98	**24.4**	0.4

*Diagnostic standard: For *atrioventricular dissociation,* ventricular-paced rhythm at a rate independent of the atrial rate.
†Definition of findings: For *varying arterial pulse*, varying amplitude of radial or carotid pulse by palpation.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant.
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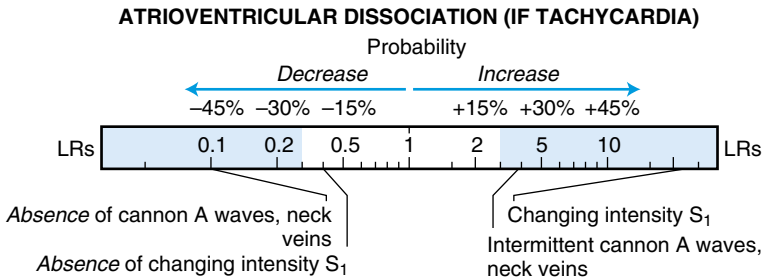


D. IRREGULAR RHYTHM THAT VARIES WITH RESPIRATION

This rhythm is sinus arrhythmia, an especially common and prominent arrhythmia in younger patients. The pulse characteristically quickens during inspiration and slows during exhalation (see Fig. 16.2).[20] The slowing during expiration is sometimes so conspicuous it mimics the finding of a pause.

E. IRREGULARLY IRREGULAR RHYTHM (CHAOTIC RHYTHM)

This term describes a cadence of ventricular and radial beats that is completely irregular and unpredictable. The diagnosis is usually atrial fibrillation. In studies of over 2000 patients, the finding of an *irregular* radial pulse increases the probability of atrial fibrillation (LR = 4.6, EBM Box 16.2), whereas the absence of this finding (i.e., the pulse is *regular*) decreases probability of atrial fibrillation (LR = 0.1). In one of these studies, using just 20 seconds of observation, the finding of a **chaotic pulse** markedly increased the probability of atrial fibrillation (LR = 24.1).

Frequent multifocal premature contractions may sometimes seem chaotic at the bedside, but two findings distinguish this rhythm from atrial fibrillation:

(1) **Venous pulse.** In atrial fibrillation, the venous pulse is simple and consists of only one wave per cardiac cycle (i.e., there is no A wave and the *x*' descent is diminished, revealing a sole *y* descent; see Chapter 36). In frequent

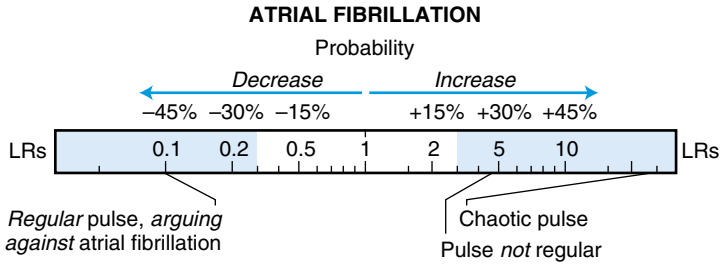
premature contractions, in contrast, the venous pulse is complex and consists of intermittent cannon A waves superimposed on two venous movements per cardiac cycle.

(2) **Rhythm of ventricular pulse** (Fig. 16.4). In atrial fibrillation, the interval between ventricular beats is random, and it is quite common to have one pause followed by an even longer pause. In frequent premature contractions,

EBM BOX 16.2
*Atrial Fibrillation**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Pulse *not* regular[21-24]	90-98	70-94	**4.6**	**0.1**
Chaotic pulse[23]	54	98	**24.1**	0.5

*Diagnostic standard: For *atrial fibrillation*, electrocardiogram.
†Definition of findings: For *chaotic pulse,* "frequent or continuous irregularity" during 20-second examination of the radial pulse.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
Click here to access calculator



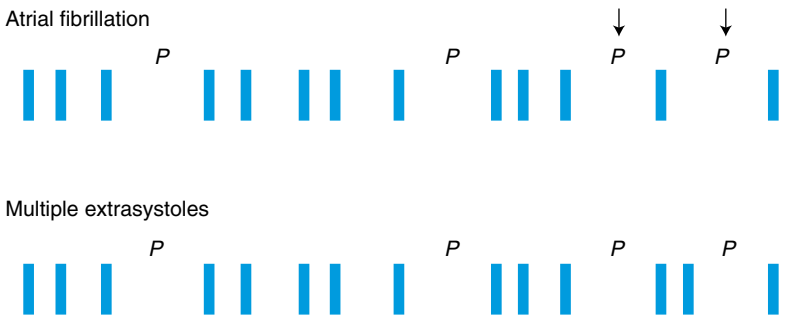


FIG. 16.4 THE CHAOTIC RHYTHM. The irregularly irregular or chaotic rhythm may represent atrial fibrillation (*top*) or sinus rhythm with multiple extrasystoles (*bottom*). "*P*" marks conspicuous pauses that appear in the cadence of *apical* heart tones. (Each bar depicts one cardiac cycle, or one *lub dup*.) In this example, the cadence of the two arrhythmias is identical until the end of the tracing: in atrial fibrillation, two pauses occur in a row (*arrows*), thus distinguishing it from the pauses of multiple extrasystoles, which are flanked by quick beats or beats of normal cadence (see the text).

this is impossible because the pause must be followed by another quick beat or the normal sinus interval. This difference in rhythm, which again focuses on the ventricular rhythm at the apex, not the radial pulse, is quite conspicuous once the clinician is aware of it.

The references for this chapter can be found on www.expertconsult.com.

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